In the third group of eighty-nine cases, the pseudo-membranous angina was more chronic in its course. It began as a pseudo-membranous angina or as a catarrhal angina, later becoming membranous, the main disease following its regular course. At the end of the first week the fever recurred, and otitis, synovitis, or erysipelas might be found, but more frequently a new swelling of the submaxillary glands developed. With this the throat, which was usually clean, became affected again within twenty-four or forty-eight hours, and symptoms of the malignant form developed, to which the child sometimes succumbed.

In all these groups the angina is due to the scarlatinal poison, and is aggravated in the last two groups by a secondary streptococcic infection. For local treatment the author advises interstitial injections of a solution of carbolic acid of 3 or 5 per cent. strength, half a Pravaz syringeful of the solution being injected twice daily into each tonsil. This treatment, which seemed of no benefit in the acute malignant cases, was, however, successful in fifty-six out of the sixty cases of the chronic form. In no case were symptoms of carbolic-acid poisoning produced by these injections.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

W. T. COUNCILMAN, M.D.,

SHATTUCK PROFESSOR OF PATHOLOGICAL ANATOMY, HARVARD UNIVERSITY.

AND

F. B. MALLORY, M.D.,

ASSISTANT PROFESSOR OF PATHOLOGICAL ANATOMY, HARVARD UNIVERSITY.

Syringomeylia.—Saxer (*Centralblatt für Allgemeine Pathologie u. Pathologische Anatomie*, 1898, Bd. ix. p. 6) says that in 1892 E. Cramer summarized in the *Centralblatt für Allgemeine Pathologie* those works which had appeared during the year 1891 on the subject of syringomyelia. The conclusions reached by him in regard to the views held at that time on the subject of syringomyelia were as follows: 1. The etiology of syringomyelia is not yet settled. In certain cases anomalies of development and injury seem to play a certain *rôle*. 2. The symptom-complex of syringomyelia can be produced by various lesions, but in most cases the lesion is a simple chronic growth of neuroglia tissue. 3. The lesion is generally localized in the gray matter behind the central canal of the cord. 4. Morvan's disease is clinically and pathologically only a form of syringomyelia. In Morvan's disease the peripheral nerves may also be diseased. 5. The disease may last two or three years (glioma) to thirty or forty years (simple growth of neuroglia tissue). 6. The cavity arises by the breaking down of pathological tissue (usually simple growth of neuroglia tissue). 7. The connection of the central canal with the cavity is secondary. In like manner Saxer reviews 146 articles which have appeared on this subject since 1892. He discusses first *hydromyelia*, in the classical cases a tube-like dilatation of the spinal canal, lined with characteristic epithelium. Variations of this form, however, occur; the epithelium may be wanting over large areas, the central canal may be double, the cavity may be more or less separated from the central canal in places, or the neuroglia tissue around the central canal may be increased. Leyden has drawn the conclusion from such cases that most syringomyelias are hydromyelias dependent on congenital anomalies. Saxer then takes up *syringomyelia*, under which term is understood an elongated cavity in the cord, which is either entirely independent of the normal central canal or is only connected with it accidentally. The pathogenesis of these cavities is various. In the opinion of most writers they arise most frequently in consequence of the breaking down of a primary growth of neuroglia tissue. To this new formation of neuroglia tissue the terms *gliosis*, *gliomatosis*, and *glioma* have been applied. K. Miura insists on a sharp distinction being made between true malignant glioma and other forms of neuroglia tissue-growth to which the term gliosis should be applied. The indefinite term "gliomatosis" should be replaced by "diffuse glioma" when it refers to an infiltrating glioma, otherwise by "gliosis." Most authors are of the opinion that in syringomyelia the growth of neuroglia tissue is primary, and the cavity secondary through the breaking down of the newly formed tissue. This view is strongly opposed both by Hoffmann and by Schlesinger in their extensive monographs on this subject. They believe that the starting point of these changes is to be found in congenital anomalies of the central canal combined with hydromyelia. This view is not entirely new, but it is now advanced with the support of the latest discoveries in the histogenesis and histology of the central nervous system, particularly the latest results in regard to the developmentof the ependyma and the neuroglia. In order to make the line of argument clear, Saxer summarizes, for the sake of the uninitiated, the discoveries of the last few years in regard to the neuroglia.

According to Hoffmann, the starting point of the disease is to be found in the majority of cases in congenital anomalies of development, as shown by groups of embryonic cells which remain behind the normal central canal along the line of closure of the neural canal. Multiplication of the central canal has a similar significance. From these embryonic remains there arises a growth of neuroglia tissue in which a cavity is usually produced by the breaking down of this newly formed tissue. Hoffmann classifies the cavities of the cord as follows:

1. Hydromyelia.
2. (*a*) Primary (central) gliosis of the cord, with or without hydromyelia.
    1. Without cavity formation.
    2. With cleft and cavity formation.

(*b*) Central gliosis with or without cleft formation, with indefinite symptoms.

The opinion of Hoffmann and Schlesinger, that syringomyelia is connected with congenital anomalies of the cord, previously had many advocates, and their views may now almost be regarded as a settled dogma. Gerlach has tried to prove that the cavity in syringomyelia is lined by connective tissue derived from the pia; but his conclusions are not generally accepted. Certain cases of tube-like cavities in the cord without gliosis have been reported by Schlesinger, Weigert, and others. Weigert claims that the consideration of syringomyelia as softening of a central gliosis has not the shadow of probability in favor of it. He calls attention to the fact that in multiple sclerosis, for instance, no breaking down and cavity formation ever take place. Hoffmann would limit the term syringomyelia strictly to cases of central gliosis, and would exclude all cases of cavity in the cord due to hemorrhage, suppuration, softening due to pressure, etc., in spite of the fact that all of the cases may show the same symptom complex.

At the end of his summary Saxer considers the connection between the syringomyelia and trauma, chronic meningitis, Morvan's disease, tabes dorsalis, and leprosy. Saxer, unfortunately, fails to summarize what he considers to be the advances made in the knowledge of syringomyelia since Cramer's review in 1892. They may, perhaps, be summed up as follows: 1. There is a tendency to separate the various forms of cavity formation in the cord as sharply as possible on an etiological basis. 2. Hoffmann would limit the term syringomyelia to cases of central gliosis. 3. The connection between most cases of syringomyelia and anomalies of development of the central canal of the cord is now generally accepted. 4. The terms *glioma* (a malignant new growth) and *gliosis* (a pathological growth of neuroglia tissue which shows none of the properties of a new growth) are clearly differentiated. The indefinite term *gliomatosis* should be given up. 5. According to Weigert the cavity in syringomyelia is primary and never due to the breaking down of a central gliosis.

Phagocytosis in Relapsing Fever.—Relapsing fever has long been considered to afford conditions most favorable for the study of phagocytosis, both because of the presence of the specific micro-organisms in the blood in large numbers, and because of the leucocytosis incident to the febrile paroxysm. These facts were early adduced by the advocates of the doctrines of phagocytosis as explaining the almost uniformly favorable outcome of the disease. Owing to the comparative rarity of relapsing fever, however, but little opportunity has been afforded for the study of these questions.

Metschnikoff, about ten years ago, after repeated failures to detect phagocytosis in the circulating blood, nevertheless found evidence of it in the spleen, and it was supposed that the germs were there overcome by the protective agencies of the body, Metschnikoff suggesting that the rapid motion of the blood in the general circulation was unfavorable to phagocytosis. To the spleen was then attributed great importance in overcoming the disease. Doubt has, however, been cast upon this theory by Tictin (1893), who found that inoculated monkeys recovered from the disease as well without the spleen as with it.

It is now found by IVANOFF (*Centralblatt für Bakteriologie u. Parasitenkunde*, 1897, xxii. 117) that phagocytosis does occur in the circulating blood in relapsing fever. In examining the blood of a patient shortly before death, Ivanoff discovered fragments of spirilla in many of the leucocytes as well as large numbers of more perfect spirilla in the plasma. The intracellular spirilla stained but feebly, and were apparently in process of disintegration.

Subsequent examination of the blood of a large number of patients suffering from relapsing fever showed similar appearances in all, and the same was true of the blood of a large number of artificially immunized monkeys.

Indeed, in these last the spirilla could be detected only in the corpuscles; none were found in the plasma, and the inference drawn by Ivanoff is that the immunity in these cases is due to heightened phagocytosis.

As before mentioned, the intracellular spirilla stain very feebly and may, consequently, be readily overlooked. For their detection a rather special method of staining is recommended: After thorough fixation on a cover by baking for an hour or more at 110° to 120° C. the specimen is immersed for from one to three minutes, with gentle warming, in a staining solution consisting of 20 to 25 c.c. of Roux's stain,[1] to which 2 to 4 c.c. of Ziehl's carbolic fuchsin have been added. The preparation is then washed in water, dried, and mounted in balsam.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

EDWARD F. WILLOUGHBY, M.D.,
OF LONDON,

AND

CHARLES HARRINGTON, M.D.,
INSTRUCTOR IN MATERIA MEDICA AND HYGIENE, HARVARD MEDICAL SCHOOL.

A New Experiment in the Treatment of Town Sewage.—The pollution of rivers with sewage and of wells with the ooze from cesspits in which the excreta are deposited at a depth at which nitrifying bacteria are no longer found has, with increasing density of population, reached a pitch on both sides of the Atlantic which cries for redress, and must no longer be tolerated. The remedy for the rural evil is simple enough: the suppression of the common privy and the substitution of earth closets or dry catches, with the immediate application of all excreta to the garden plot in the case of cottages, and in that of large establishments the collection of all excreta, water-closet, and domestic slops in perfectly impervious cemented tanks, with pumps and arrangements for the regular disposal of the liquid by hose or irrigation over garden, orchard, or meadow.

Town sewage must ultimately return to the rivers whence it was drawn, or toward which it was wending when impounded or pumped for the public water-supply; but the great problem of the day is how to purify it so far as not sensibly to pollute the stream. Chemical processes stand *de facto* condemned as unscientific and opposed to the elementary principles of economics. Even if the chemicals added are not such as to render the effluent hurtful to animal and vegetable life, unfit, therefore, for discharge into rivers or distribution over the land, they act by destroying the microbes whose function it is to transmute offensive organic matter into purely inorganic salts, to mineralize it as the process has been called, and so to render it capa-

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[1] Roux's stain is prepared by mixing 15 c.c. of a 1 per cent. aqueous solution of dahlia, 45 c.c. of a 1 per cent. aqueous solution of methylene green, and 10 drops of formalin.